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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/900,700	07/06/2001	Keith D. Allen	R-616	3949

7590 10/23/2002

DELTAGEN, INC.
1003 Hamilton Avenue
Menlo Park, CA 94025

EXAMINER
PARAS JR, PETER

ART UNIT	PAPER NUMBER
1632	13

DATE MAILED: 10/23/2002

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Please find below and/or attached an Office communication concerning this application or proceeding.

RESPONSE DUE 23-JAN-02

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Office Action Summary

Application No.	Applicant(s)	
09/900,700	ALLEN, KEITH D.	
Examiner	**Art Unit**	
Peter Paras, Jr.	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on *09 October 2002* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

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4)☒ Claim(s) *1-23* is/are pending in the application.

4a) Of the above claim(s) *1-7,9,11-16 and 23* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *8,10 and 17-22* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *06 July 2001* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) *3* .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

DETAILED ACTION

Claims 1-23 are pending.

Information Disclosure Statement

The IDS filed on 10/29/01 has been considered. It appears from the file wrapper of the instant application that there may have been an additional IDS submitted on or around 11/28/01 as Paper No: 4. However, the additional IDS has not been found by the Examiner and cannot be considered at this time. If the additional IDS becomes available or is resubmitted by Applicants then it will be considered by the Examiner.

Specification

The Brief Description of the Drawings in the instant specification is objected to because there is no description of Figure 2A.

Sequence Compliance

This application contains sequence disclosures that are encompassed by the definitions for nucleotide and/or amino acid sequences set forth in 37CFR 1.821(a)(1) and (a)(2). However, this application fails to comply with the requirements of 37 CFR 1.821 through 1.825 for the reason(s) set forth on the attached **N**otice To Comply With Requirements For Patent Applications Containing **N**ucleotide Sequence And/Or Amino Acid Sequence Disclosures. Figure 2A comprises an unidentified sequence.

Applicants are required to comply with all of the requirements of 37 C.F.R. §§ 1.821 through 1.825. *Any* response to this Office Action, which fails to meet all of these

requirements, will be considered non-responsive. The nature of the noncompliance with the requirements of 37 C.F.R. §§ 1.821 through 1.825 did not preclude the examination of the application on the merits, the results of which are communicated below.

Election/Restrictions

Applicant's election with traverse of Group III, claims 8,10, and 17-22, in Paper No. 12 is acknowledged. The traversal is on the ground(s) that the Examiner has not shown that a serious burden would be required to examine all the claims. This is not found persuasive because each of the Inventions requires a separate search status. In particular, it is maintained that the products of Groups I, II, III, VI and VII are different each from the other; they each have different chemical structures and can be used in materially different methods that require different technical considerations. For example, the DNA targeting construct of Group I can be used to disrupt a CRFR2 gene in a somatic cell *in vitro*, The cells of Group II can be used to produce and isolate a protein *in vitro*, the transgenic non-human animal of Group III can be used as a model of disease, the unknown agent of Group VI can be used for modulating the expression of CRFR2 in a somatic cell *in vitro*, and the phenotypic data of Group VII can be used for statistical analysis with a computer. It is maintained that the products of Inventions I, II, III, VI and VII are distinct due to their divergent subject matter (DNA targeting construct, cell s, transgenic non-human animal, unknown agent that can modulate the expression of a CRFR2, and data in an electronic database) and are separately classified and searched.

It is maintained that the methods of Groups IV and V are distinct, comprising different methodologies and using different products. For example, the method of Group V can be practiced in a somatic cell *in vitro*, while the method of Group IV is required to be practiced in a transgenic non-human animal. It is maintained that the methods of Groups IV and V are distinct as they are directed to different methods that require the use of different products that need different technical considerations (transgenic non-human animals and somatic cells *in vitro*) and are separately searched and classified.

It is maintained that the products of Groups I, II, III, VI and VII are distinct from the methods of Groups IV and V; the products of Groups I, II, III, VI and VII can be used in methods, which require different reagents and technical considerations from the methods of Groups IV and V. For example, the DNA targeting construct of Group I may be used as a probe in a hybridization assay *in vitro* while the transgenic non-human animal of Group III may be used to produce antibodies to an antigen, the cells of Group II can be used to produce a protein *in vitro*, while the method of Group V may be used to identify agents that modulate the expression of a CRFR2. The method of Group IV may be practiced with agents that have different chemical structures from the agent of Group VI. It is maintained that the products of Groups I, II, III, VI, and VII are distinct from and can be used in different methods (hybridization assays, generating antibodies, producing a protein) from the screening methods of Groups IV and V.

Therefore it is maintained that all the inventions are distinct each from the other for the reasons given above. The requirement is still deemed proper and is therefore made FINAL.

Please note that after a final requirement for restriction, the Applicants, in addition to making any response due on the remainder of the action, may petition the Commissioner to review the requirement. Petition may be deferred until after final action on or allowance of claims to the invention elected, but must be filed not later than appeal. A petition will not be considered if reconsideration of the requirement was not requested. (See § 1.181.).

Claims 1-7, 9, 11-16, and 23 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected Invention, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in Paper No. 8.

Claim Rejections - 35 USC § 112, 1st paragraph

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 8, 10, and 17-22 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a transgenic mouse whose genome comprises a homozygous disruption of the nucleotide sequence set forth in SEQ ID NO:

1, wherein said nucleotide sequence encodes a CRFR2, wherein the mouse exhibits a phenotype of decreased activity, hypoactivity, and a decreased susceptibility seizure, and a method of making the same transgenic mouse comprising introducing a targeting construct into an ES cell, introducing the ES cell into a blastocyst, and implanting the blastocyst into a pseudopregnant mouse, and allowing said blastocyst to develop to term, does not reasonably provide enablement for all other transgenic non-human animals and methods of making transgenic mice embraced by the claims. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The claims are directed transgenic non-human animals, particularly a mouse, that comprise a disruption in a CRFR2 gene, the sequence of which is set forth in SEQ ID NO: 1, wherein the mouse exhibits a phenotype of decreased activity, hypoactivity, and decreased susceptibility to seizures. The claims are further directed a method of producing a transgenic mouse comprising a disruption in a CRFR2 gene.

The specification teaches the generation of transgenic mice by disruption of the nucleotide sequence set forth in SEQ ID NO: 1, wherein SEQ ID NO: 1 encodes a CRFR2. See page 6, at lines 11-17, page 9 at lines 15-24, and the working example on pages 53-54, of the specification. The specification teaches that transgenic mice whose genome comprises a homozygous disruption in SEQ ID NO:1 exhibit a phenotype of decreased activity, hypoactivity, and decreased susceptibility to seizures in response to metrazol as compared to wild-type mice, as a result of the disruption of the nucleotide

sequence set forth in SEQ ID NO: 1. See pages 53-54 of the specification. While the specification has taught the generation of such a transgenic knockout mouse having a phenotype of decreased activity, hypoactivity, and decreased susceptibility to seizures, the specification has not taught the generation of the other transgenic non-human animals encompassed by the claims. The specification has also taught a method of producing a transgenic mouse comprising a disruption in a CRFR2 gene as set forth in the nucleotide sequence set forth in SEQ ID NO: 1, wherein the method requires introduction of a targeting construct into an embryonic stem cell. The specification has not taught how to create a transgenic mouse comprising a disruption in a CRFR2 gene wherein a targeting construct is introduced into any other cell. The working examples, guidance and relevant teachings provided by the instant specification are directed to the creation of the above transgenic mouse but do not support the creation of other transgenic non-human animals encompassed by the claims. See pages 53-54.

With regard to claim breadth, the standard under 112, first paragraph entails the determination of what the claims recite and what the claims mean as a whole. In addition, when analyzing the enabled scope of the claims, the teachings of the specification are taken into account because the claims are to be given their broadest reasonable interpretation that is consistent with the specification. As such, in light of the specification, the claimed invention is properly interpreted with regard to the disclosed phenotype of the exemplified transgenic mouse whose genome comprises a homozygous disruption of the nucleotide sequence set forth in SEQ ID NO: 1. Such an interpretation is consistent with the specification despite that the claimed non-human

animals require only that they comprise a disrupted CRFR2, particularly the nucleotide sequence set forth in SEQ ID NO: 1. This is because, with regard to the enablement requirement, one of skill in the art must be provided with both how to make and use the claimed invention. As such, the enabled scope of the claimed invention, in light of the teachings of the specification, is found to be the generation of a transgenic mouse whose genome comprises a homozygous disruption of the nucleotide sequence set forth in SEQ ID NO: 1 which exhibit a phenotype of decreased activity, hypoactivity, and decreased susceptibility to seizures.

The following aspect of the rejection under 35 U.S.C. 112, first paragraph is directed to claims 8, 10 and 17-22 as they read on transgenic knockout non-human animals, use of embryonic stem cells to make a transgenic mouse, and germline transmission of ES cells:

Both the specification and the state of the art have taught that the transgenic knockout technology requires the use of embryonic stem cells that have been genetically manipulated to comprise a disruption in a nucleotide sequence of interest. The specification has not taught creation of a transgenic knockout non-human animal by methods that do not require embryonic stem cells. Presently, the transgenic knockout technology is limited to the mouse system. See below.

With regard to the claim breadth directed to transgenic non-human animals, the specification fails to teach the production of any transgenic non-human animal comprising a disruption in a CRFR2 gene other than a transgenic knockout mouse whose genome comprises a homozygous disruption in the nucleotide sequence set

forth in SEQ ID NO: 1. It is well known in the knockout art that the production of knockout animals other than mice is undeveloped. This is because ES cell technology is generally limited to the mouse system, at present, and that only "putative" ES cells exist for other species. See Moreadith et al. at page 214, Summary. Seamark (Reproductive Fertility and Development, 1994) supports this observation by reporting that totipotency for ES cell technology in many livestock species has not been demonstrated (page 6, Abstract). Likewise, Mullins et al (Journal of Clinical Investigation, 1996) state that "although to date chimeric animals have been generated from several species including the pig, in no species other than the mouse has germline transmission of an ES cell been successfully demonstrated." (page S38, column 1, first paragraph). Moreover, with regard to claim 10 neither the state of the art nor the prior art of record has provided guidance for use of cells, other than ES cells for production of a transgenic knockout mouse. It would be unpredictable if other cells could be used for the production of a transgenic knockout mouse because other cells may be not totipotent or transmit through the germline as ES cells do. Even more, claims 8 and 17-22 as written do not appear to require germline transmission of the disrupted nucleotide sequence. These claims may be broadly interpreted to read on a single cell comprising a disrupted nucleotide sequence. Since the claims do not require germline transmission of the disrupted nucleotide sequence it would be unpredictable if an ES cell comprises the disrupted nucleotide sequence. As stated above the evidence of record does not support germline transmission of non-ES cells. Also, it would be unpredictable if a disruption of a nucleotide sequence in a single cell would result in a phenotype; the

instant specification has not provided any uses for a transgenic mouse that does not exhibit a phenotype resulting from disruption of a nucleotide sequence (see below). As the claims are directed to transgenic non-human animals (claim 8) or a method that requires the use of a cell to in the production of a transgenic mouse (claim 10), wherein the cell is interpreted to read on an embryonic stem cell (as in claim 10) comprising a disruption in a CRFR2 gene, which must be generated by the introduction of a transgene into an ES cell or transgenic non-human animals, particularly a mouse, that do not exhibit germline transmission of a disrupted nucleotide sequence, the state of the art supports that only mouse ES cells were available for use for production of transgenic mice whose genomes comprise a homozygous disruption of a CRFR2 gene as set forth in SEQ ID NO: 1. Given the unpredictable state of the art it would have required undue experimentation for the skilled artisan to create transgenic knockout non-human animals of species other than the mouse or to make a transgenic knockout mouse with a cell other than an embryonic stem cell.

Claims 8 and 17 encompass transgenic non-human animals, particularly a mouse, that comprise a disruption in a CRFR2 gene, particularly the nucleotide sequence set forth in SEQ ID NO: 1, that do not exhibit any particular phenotype. The state of the art at the time of filing was such that one of skill could not predict the phenotype of a knockout mouse (Moreadith et al., 1997, J. Mol. Med., Vol. 75, pages 208-216; see page 208, column 2, last full paragraph). Moens et al. (Development, Vol. 119, pages 485-499, 1993) disclose that two mutations produced by homologous recombination in two different locations of the N-myc gene produce two different

phenotypes in mouse embryonic stem cells, one leaky and one null (see abstract). The specification has asserted that the nucleotide sequence set forth in SEQ ID NO: 1 encodes a CRFR2. However, it would be difficult to predict any phenotype resulting from disruption of the sequence of SEQ ID NO: 1 in light of the above. The specification discloses a phenotype exhibited by knockout mice comprising a disruption in the nucleotide sequence set forth in SEQ ID NO: 1 is decreased activity, hypoactivity, and decreased susceptibility to seizures. See pages 53-54 of the specification. Claims 8 and 17, as written, do not include a phenotype that differs from the wild-type mouse. Moreover the skilled artisan would not know how to use a transgenic knockout non-human animal that lacks a phenotype, particularly because the instant specification has not provided uses for such; the transgenic mice that have a phenotype may be used for drug testing according to the instant specification. The specification overcomes the unpredictability in obtaining a phenotype associated with a disruption of the nucleotide sequence set forth in SEQ ID NO: 1, which is asserted to encode a CRFR2; however, the claims are not commensurate in scope with the enabled phenotype disclosed in the specification. Inclusion of a phenotype associated with a disruption of the nucleotide sequence set forth in SEQ ID NO: 1 or a CRFR2 gene in a mouse in the claims would overcome this aspect of the rejection. Given the unpredictable nature of a phenotype that results from disruption of a nucleotide sequence it would have required undue experimentation for the skilled artisan to use a transgenic non-human knockout animal that lacks a phenotype.

As a final issue, claims 17-22 encompass transgenic mice comprising a disruption in a homolog of the nucleotide sequence set forth in SEQ ID NO: 1. The specification has disclosed mice that comprise a disruption in the nucleotide sequence set forth in SEQ ID NO: 1, while the specification has not taught mice comprising a disruption in a homolog of the nucleotide sequence set forth in SEQ ID NO: 1. The claims broadly encompass disruption of nucleotide sequences that are homologs of the nucleotide sequence set forth in SEQ ID NO: 1, which have different structures from the nucleotide sequence set forth in SEQ ID NO: 1; given the structural differences it may presumed that the encoded proteins possess different functions. Moreover, since the claims broadly encompass disrupting homologs of SEQ ID NO: 1, the members of the genus of such homologs may possess different functions and chemical structures, it would be unpredictable if disrupting homologs of SEQ ID NO: 1, would result in the phenotype of decreased activity, hypoactivity, and decreased susceptibility to seizures as exhibited by transgenic mouse exemplified in the working example on pages 53-54 of the specification; the specification has not disclosed any homologs of the nucleotide sequence set forth in SEQ ID NO: 1. The issue of the unpredictability of a phenotype resulting from disruption of a homolog of SEQ ID NO: 1 arises because the state of the art at the time of filing was such that one of skill could not predict the phenotype of a knockout mouse as discussed above. See Moreadith. Moens et al. (see above) disclose that two mutations produced by homologous recombination in two different locations of the N-myc gene produce two different phenotypes in mouse embryonic stem cells, one leaky and one null (see abstract). The specification has asserted that

the nucleotide sequence set forth in SEQ ID NO: 1 encodes a CRFR2 but has not provided any teachings with regard to homologs of the nucleotide sequence set forth in SEQ ID NO: 1. It would be difficult to predict any phenotype resulting from disruption of a homolog of the nucleotide sequence of SEQ ID NO: 1 in light of the above. The specification discloses a phenotype exhibited by knockout mice comprising a disruption in the nucleotide sequence set forth in SEQ ID NO: 1 is decreased activity, hypoactivity, and decreased susceptibility to seizures but has not disclosed a phenotype resulting from the disruption of a homolog of SEQ ID NO: 1. As such it would have required undue experimentation for the skilled artisan to make and use a transgenic mouse comprising a disruption of a homolog of the nucleotide sequence set forth in SEQ ID NO: 1 without a reasonable expectation of success.

Therefore, in view of the quantity of experimentation necessary to determine the parameters listed above for the production of transgenic non-human animals comprising a disruption in a CRFR2 gene, the lack of direction or guidance provided by the specification for the production of transgenic non-human animals comprising a disruption in a CRFR2 gene, the absence of working examples for the demonstration or correlation to the production of a transgenic knockout non-human animal that exhibits a phenotype other than the exemplified mouse, the unpredictable state of the art with respect to a phenotype that results from disruption of a given nucleotide sequence, the undeveloped art pertaining to the establishment of true embryonic stem (ES) cells of animal species other than mouse, and the breadth of the claims drawn to all non-human animals and to homologs of the nucleotide sequence set forth in SEQ ID NO: 1, it would

have required undue experimentation for one skilled in the art to make and/or use the claimed invention.

It is noted that the following claim language may be sufficient to overcome the preceding enablement rejection: A transgenic mouse whose genome comprises a homozygous disruption of a CRFR2 gene, the nucleotide sequence of which is set forth in SEQ ID NO: 1, exhibiting a phenotype of decreased activity, hypoactivity, and decreased susceptibility to seizures.

Claims 17-22 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The claims are directed transgenic non-human animals, particularly a mouse, that comprise a disruption in the nucleotide sequence set forth in SEQ ID NO: 1 or a homolog thereof, wherein the mouse exhibits a phenotype of decreased activity, hypoactivity, and decreased susceptibility to seizures.

Vas-Cath Inc. v. Mahurkar, 19USPQ2d 1111 (Fed. Cir. 1991), clearly states that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession *of the invention*. The invention is, for purposes of the 'written description' inquiry, *whatever is now claimed.*" Vas-Cath Inc. v. Mahurkar, 19USPQ2d at 1117. The specification does not "clearly allow persons of

ordinary skill in the art to recognize that [he or she] invented what is claimed." Vas-Cath Inc. v. Mahurkar, 19USPQ2d at 1116.

The specification has provided a description for the nucleotide sequence set forth in SEQ ID NO: 1. The specification has asserted that the nucleotide sequence set forth in SEQ ID NO: 1 encodes a CRFR2. However, the nucleotide sequences that homologs of the nucleotide sequence set forth in SEQ ID NO: 1 have not been disclosed. Based upon the prior art there is expected to be variation among the species of polynucleotides that comprise the genus of nucleotide sequences as set forth in SEQ ID NO: 1. The specification has failed to disclose the nucleotide sequences of any nucleic acid that are homologs of the nucleotide sequence set forth in SEQ ID NO: 1. There is no evidence on the record of a relationship between the structures of any DNA molecules, which are homlogs of the nucleotide sequence set forth in SEQ ID NO: 1, that would provide any reliable information about the structures of other such DNA molecules. There is no evidence on the record that the nucleotide sequences that are homologs of the nucleotide sequence set forth in SEQ ID NO: 1 had a known structural relationship to other DNA sequences encompassed within the genus. Furthermore, the evidence of record has not provided evidence of a structural relationship between the nucleotide sequence set forth in SEQ ID NO: 1 and the nucleotide sequences that homologs of the nucleotide sequence set forth in SEQ ID NO: 1. Moreover it is not known if the homologs of SEQ ID NO: 1 would encode proteins that would even possess the biological activity of the protein encoded by the nucleotide sequence set forth in SEQ ID NO: 1. The claimed invention as a whole is not adequately described if

the claims require essential or critical elements which are not adequately described in the specification and which is not conventional in the art as of applicants effective filing date. Possession may be shown by actual reduction to practice, clear depiction of the invention in a detailed drawing, or by describing the invention with sufficient relevant identifying characteristics such that a person skilled in the art would recognize that the inventor had possession of the claimed invention. Pfaff v. Wells Electronics, Inc., 48 USPQ2d 1641, 1646 (1998).

In the instant case the claimed embodiments of nucleotide sequence that are homologs of the nucleotide sequence set forth in SEQ ID NO: 1 lack a written description. The specification fails to describe what DNA molecules fall into this genus. The skilled artisan cannot envision the detailed chemical structure of the encompassed DNA molecules that are homologs of the nucleotide sequence forth in SEQ ID NO: 1, and therefore conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method of isolating it. See *Fiers v. Revel*, 25 USPQ2d 1601, 1606 (Fed. Cir. 1993) and *Amgen Inc. v. Chugai Pharmaceutical Co. Ltd.*, 18 USPQ2d 1016 (Fed. Cir. 1991).

One cannot describe what one has not conceived. See *Fiddes v. Baird*, 30 USPQ2d 1481, 1483. In *Fiddes*, claims directed to mammalian FGF's were found to be unpatentable due to lack of written description for that broad class. The specification provided only the bovine sequence.

In view of the above considerations one of skill in the art would not recognize that applicant was in possession of the necessary common features or attributes possessed by member of the genus of nucleotide molecules that are homologs of the nucleotide sequence forth in SEQ ID NO: 1. Moreover, the art would generally recognize that there would be variation among the species of the genus of polynucleotide molecules that are homologs of the nucleotide sequence set forth in SEQ ID NO: 1. Therefore, Applicant was not in possession of the genus of nucleotide molecules that are homologs of the nucleotide sequence set forth in SEQ ID NO: 1 as encompassed by the claims. University of California v. Eli Lilly and Co., 43 USPQ2d 1398, 1404, 1405 held that to fulfill the written description requirement, a patent specification must describe an invention and do so in sufficient detail that one skilled in the art can clearly conclude that "the inventor invented the claimed invention."

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.
>
> (e) the invention was described in-
> (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effect under this subsection of a national application published under section 122(b) only if the international application designating the United States was published under Article 21(2)(a) of such treaty in the English language; or
> (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that a patent shall not be deemed filed in the United States for the purposes of this subsection based on the filing of an international application filed under the treaty defined in section 351(a).

Claims 8 and 10 are rejected under 35 U.S.C. 102(a) as being anticipated by Coste et al (Nature Genetics, 2000, 24: 403-409).

The claims are directed to a transgenic non-human animal comprising a disruption in a CRFR2 gene and a method for producing a transgenic mouse comprising a disruption in a CRFR2 gene.

For the purposes of the this rejection a CRFR2 gene is interpreted to be a CRHR2. This interpretation has been made because the prior art as set forth in the specification on pages 1-4 sets forth that a CRFR2 gene and a CRHR2 gene are the same. The difference being in name only, corticotropin-releasing factor receptor as opposed to corticotropin-releasing hormone receptor.

Coste et al teach a transgenic mouse comprising a disruption in the CRHR2 gene. Coste et al teach that the transgenic mouse is created by introducing a targeting vector into ES cells, transferring the ES cells to a blastocyst and then implanting the blastocyst into a pseudopregnant female mouse, wherein said female mouse gives birth to a chimeric mouse, and breeding said chimeric mouse to produce the transgenic mouse. See page 403, column 1, first paragraph as well as the Materials and Methods section on pages 406-408.

Thus, the teachings of Coste et al anticipate all of the instant claim limitations.

Claims 8 and 10 are rejected under 35 U.S.C. 102(a) as being anticipated by Bale et al (Nature Genetics, 2000, 24: 410-414).

The claims are directed to a transgenic non-human animal comprising a disruption in a CRFR2 gene and a method for producing a transgenic mouse comprising a disruption in a CRFR2 gene.

For the purposes of the this rejection a CRFR2 gene is interpreted to be a CRHR2. This interpretation has been made because the prior art as set forth in the specification on pages 1-4 sets forth that a CRFR2 gene and a CRHR2 gene are the same. The difference being in name only, corticotropin-releasing factor receptor as opposed to corticotropin-releasing hormone receptor.

Bale et al teach a transgenic mouse comprising a disruption in the CRHR2 gene. Coste et al teach that the transgenic mouse is created by introducing a targeting vector into ES cells, transferring the ES cells to a blastocyst and then implanting the blastocyst into a pseudopregnant female mouse, wherein said female mouse gives birth to a chimeric mouse, and breeding said chimeric mouse to produce the transgenic mouse. See page 410, column 1, first paragraph bridging to page 411 as well as the Materials and Methods section on page 412.

Thus, the teachings of Bale et al anticipate all of the instant claim limitations.

Claims 8 and 10 are rejected under 35 U.S.C. 102(a) as being anticipated by Kishimoto et al (Nature Genetics, 2000, 24: 415-419).

The claims are directed to a transgenic non-human animal comprising a disruption in a CRFR2 gene and a method for producing a transgenic mouse comprising a disruption in a CRFR2 gene.

For the purposes of the this rejection a CRFR2 gene is interpreted to be a CRHR2. This interpretation has been made because the prior art as set forth in the specification on pages 1-4 sets forth that a CRFR2 gene and a CRHR2 gene are the same. The difference being in name only, corticotropin-releasing factor receptor as opposed to corticotropin-releasing hormone receptor.

Kishimoto et al teach a transgenic mouse comprising a disruption in the CRHR2 gene. Kishimoto et al teach that the transgenic mouse is created by introducing a targeting vector into ES cells, transferring the ES cells to a blastocyst and then implanting the blastocyst into a pseudopregnant female mouse, wherein said female mouse gives birth to a chimeric mouse, and breeding said chimeric mouse to produce the transgenic mouse. See page 415, column 1, first paragraph, Figure 1 on page 415 as well as the Materials and Methods section on pages 418.

Thus, the teachings of Kishimoto et al anticipate all of the instant claim limitations.

Claims 8 and 10 are rejected under 35 U.S.C. 102(e) as being anticipated by Lee et al (US 6,353,152; effective filing date of 7/15/1999).

The claims are directed to a transgenic non-human animal comprising a disruption in a CRFR2 gene and a method for producing a transgenic mouse comprising a disruption in a CRFR2 gene.

Lee et al teach a transgenic mouse comprising a disruption in the CRHF2 gene. Lee et al teach that the transgenic mouse is created by introducing a targeting vector into ES cells, transferring the ES cells to a blastocyst and then implanting the blastocyst into a pseudopregnant female mouse, wherein said female mouse gives birth to a chimeric mouse, and breeding said chimeric mouse to produce the transgenic mouse. See column 8 and throughout entire document.

Thus, the teachings of Lee et al anticipate all of the instant claim limitations.

Conclusion

No claim is allowed. Claims 17-22 appear to be free of the prior art of record but are subject to other rejections.

Any inquiry concerning this communication or earlier communications from the examiner(s) should be directed to Peter Paras, Jr., whose telephone number is 703-308-8340. The examiner can normally be reached Monday-Friday from 8:30 to 4:30 (Eastern time).

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Deborah Reynolds, can be reached at 703-305-4051. Papers related to this application may be submitted by facsimile transmission. Papers should be faxed via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center numbers are (703) 308-4242 and (703) 305-3014.

Inquiries of a general nature or relating to the status of the application should be directed to Patsy Zimmerman whose telephone number is (703) 308-0009.

Peter Paras, Jr.

Art Unit 1632

Pete Paras
Art Unit 1632

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INFORMATION DISCLOSURE CITATION
PTO-1449

Atty Docket: R-616	Serial No.: 09/900,700
TRANSGENIC MICE CONTAINING CRFR2 CORTICOTROPIN-RELEASING FACTOR RECEPTOR GENE DISRUPTIONS	Applicant: ALLEN
Date: October 29, 2001	Filing Date: July 6, 2001 / Group Art Unit: Unassigned

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U.S. PATENT DOCUMENTS

EXAMINER'S INITIALS	PATENT NO.	DATE	NAME	CLASS	SUBCLASS	FILING DATE

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FOREIGN PATENT DOCUMENTS

EXAMINER'S INITIALS	PATENT NO.	DATE	COUNTRY	CLASS	SUBCLASS	Translation Yes	Translation No

OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)

PP	Bale, T. L. et al., Nature Genetics, 24:410-414 (2000), "Mice deficient for corticotropin-releasing hormone receptor-2 display anxiety-like behaviour and are hypersensitive to stress"
	Berridge, C. W. et al., Pharm. Biochem. Behav., 34:517-519 (1989), "CRF and Restraint-Stress Decrease Exploratory Behavior in Hypophysectomized Mice"
	Britton, D. R. et al., Life Sci., 38:211-216 (1986), "Dexamethasone Suppresses Pituitary-Adrenal But Not Behavioral Effects of Centrally Administered CRF"
	Britton, K. T. et al., Life Sci., 39:1281-1286 (1986), "Activating and 'Anxiogenic' Effects of Corticotropin Releasing Factor are not Inhibited by Blockade of the Pituitary-Adrenal System with Dexamethasone"
	Brown, M. R. et al., Brain Res., 280:75-79 (1983), "Central Nervous System Effects of Corticotropin Releasing Factor in the Dog"
	Butler, P. D. et al., J. Neurosci., 10:176-183 (1990), "Corticotropin-Releasing Factor Produces Fear-Enhancing and Behavioral Activating Effects Following Infusion into the Locus Coeruleus"
PP	Coste, S. C. et al., Nature Genetics, 24:403-409 (2000), "Abnormal adaptations to stress and impaired cardiovascular function in mice lacking corticotropin-releasing hormone receptor-2"

EXAMINER	DATE CONSIDERED
Pete Paras	10/20/07

EXAMINER: Initial if reference considered, whether or not citation is in conformance with MPEP 609; draw line through citation if not in conformance and not considered. Include copy of this form with next communication to applicant.

INFORMATION DISCLOSURE CITATION
PTO-1449

TRANSGENIC MICE CONTAINING CRFR2 CORTICOTROPIN-RELEASING FACTOR RECEPTOR GENE DISRUPTIONS

Date: **October 29, 2001**

PATENT & TRADEMARK OFFICE OCT 29 2001 JC138

Atty Docket:	Serial No.:
R-616	**09/900,700**
Applicant:	
ALLEN	
Filing Date:	**Group Art Unit:**
July 6, 2001	**Unassigned**

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EXAMINER'S INITIALS	PATENT NO.	DATE	COUNTRY	CLASS	SUBCLASS	Translation Yes	Translation No

OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)

PP	Fabbri, A. et al., Endocrinology, 127:1541-1543 (1990), "Corticotropin-Releasing Factor is Produced by Rat Leydig Cells and has a Major Local Antireproductive Role in the Testis"
	Holsboer, F. et al., N. Engl. J. Med., 311:1127-1128 (1984), "Blunted Corticotropin and Normal Cortisol Response to Human Corticotropin-Releasing Factor in Depression"
	Hsu and Hsueh, Nature Medicine, 7:605-611 (2001), "Human stresscopin and stresscopin-related peptide are selective ligands for the type 2 corticotropin-releasing hormone receptor"
	Kaye, W. H. et al., J. Clin. Endocrinol. Metab., 64:203-208 (1987), "Elevated Cerebrospinal Fluid Levels of Immunoreactive Corticotropin-Releasing Hormone in Anorexia Nervosa: Relation to State of Nutrition, Adrenal Function, and Intensity of Depression"
	Kishimoto, T. et al., Proc. Natl. Acad. Sci. USA, 92:1108-1112 (1995), "A sauvagine/corticotropin-releasing factor receptor expressed in heart and skeletal muscle"
	Kishimoto, T. et al., Nature Genetics, 24:415-419 (2000), "Deletion of *Crhr2* reveals an anxiolytic role for corticotropin-releasing hormone receptor-2"
PP	Kling, M. A. et al., J. Clin. Endocrinol. Metab., 72:260-271 (1991), "Cerebrospinal Fluid Immunoreactive Corticotropin-Releasing Hormone and Adrenocorticotropin Secretion in Cushing's Disease and Major Depression: Potential Clinical Implications"

EXAMINER	DATE CONSIDERED
Pete Thwang	10/22/08

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CLOSURE CITATION PTO-1449		Atty Docket: R-616	Serial No.: 09/900,700
IC MICE CONTAINING CRFR2 ICOTROPIN-RELEASING FACTOR RECEPTOR GENE DISRUPTIONS		Applicant: ALLEN	
Date: October 29, 2001		Filing Date: July 6, 2001	Group Art Unit: Unassigned

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FOREIGN PATENT DOCUMENTS

EXAMINER'S INITIALS	PATENT NO.	DATE	COUNTRY	CLASS	SUBCLASS	Translation Yes	Translation No

OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)

PP	Kostich, W. A. et al., Molec. Endocrinol., 12(8):1077-1085 (1998), "Molecular Identification and Analysis of a Novel Human Corticotropin-Releasing Factor (CRF) Receptor: The CRF2γ Receptor"
	Lesh, J. S. et al., Mammalian Genome, 8:944-945 (1997), "Mapping of the mouse corticotropin-releasing hormone receptor 2 gene *(Crhr2)* to Chromosome 6"
	Liaw, C. W. et al., Endocrinology, 137:72-77 (1996), "Cloning and Characterization of the Human Corticotropin-Releasing Factor-2 Receptor Complementary Deoxyribonucleic Acid"
	Meyer, A. H. et al., Genomics, 40:189-190 (1997), "Localization of the Human CRF2 Receptor to 7p21-p15 by Radiation Hybrid Mapping and FISH Analysis"
	Morley, J. E. et al., Life Sci., 31:1459-1464 (1982), "Corticotropin Releasing Factor, Grooming and Ingestive Behavior"
	Nemeroff, Charles B., Pharmacpsychiat., 21:76-82 (1988), "The Role of Corticotropin-Releasing Factor in the Pathogenesis of Major Depression"
PP	Nemeroff, C. B. et al., Science, 226:1342-1344 (1984), "Elevated Concentrations of CSF Corticotropin-Releasing Factor-Like Immunoreactivity in Depressed Patients"

EXAMINER Pete Ruoni	DATE CONSIDERED 10/25/02

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INFORMATION DISCLOSURE CITATION
PTO-1449

TRANSGENIC MICE CONTAINING CRFR2 CORTICOTROPIN-RELEASING FACTOR RECEPTOR GENE DISRUPTIONS	Atty Docket: R-616	Serial No.: 09/900,700
Date: October 29, 2001	Applicant: ALLEN	
	Filing Date: July 6, 2001	Group Art Unit: Unassigned

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EXAMINER'S INITIALS	PATENT NO.	DATE	COUNTRY	CLASS	SUBCLASS	Translation Yes	Translation No

OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)

DP	Roy-Byrne, P. P., Am. J. Psychiatry, 143:896-899 (1987), "The Corticotropin-Releasing Hormone Stimulation Test in Patients With Panic Disorder"
	Sherman, J. E. et al., Pharm. Biochem. Behav., 26:699-703 (1987), "The Effects of ICV-CRH on Novelty-Induced Behavior"
	Sirinathsinghji, D. J. et al., Nature, 305:232-235 (1983), "Corticotropin-releasing factor is a potent inhibitor of sexual receptivity in the female rat"
	Stephens, R. L., Jr. et al., Peptides, 9:1067-1070 (1988), "Intracisternal Injection of CRF Antagonist Blocks Surgical Stress-Induced Inhibition of Gastric Secretion in the Rat"
	Suda, T. et al., J. Clin. Endocrinol. Metab., 58:919-924 (1984), "Immunoreactive Corticotropin and Corticotropin-Releasing Factor in Human Hypothalamus, Adrenal, Lung Cancer, and Pheochromocytoma"
	Sutton, R. E. et al., Nature, 297:331-333 (1982), "Corticotropin releasing factor produces behavioural activation in rats"
DP	Swanson, L. W. et al., Neuroendocrinology, 36:165-186 (1983), "Organization of Ovine Corticotropin-Releasing Factor Immunoreactive Cells and Fibers in the Rat Brain: An Immunohistochemical Study"

EXAMINER	DATE CONSIDERED
Peter Bruno	10/20/03

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Notice of References Cited

Application/Control No.	Applicant(s)/Patent Under Reexamination	
09/900,700	ALLEN, KEITH D.	
Examiner	Art Unit	Page 1 of 1
Peter Paras, Jr.	1632	

U.S. PATENT DOCUMENTS

*		Document Number Country Code-Number-Kind Code	Date MM-YYYY	Name	Classification
	A	US-6353152 B1	03-2002	Lee et al	800/18
	B	US-			
	C	US-			
	D	US-			
	E	US-			
	F	US-			
	G	US-			
	H	US-			
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	N					
	O					
	P					
	Q					
	R					
	S					
	T					

NON-PATENT DOCUMENTS

*		Include as applicable: Author, Title Date, Publisher, Edition or Volume, Pertinent Pages)
	U	Moens et al. Defects in heart and lung development in compound heterozygotes for two different targeted mutations at the N-myc locus. Development, 1993, 119: 485-499.
	V	Moreadith et al. Gene targeting in embryonic stem cells: the new physiology and metabolism. J. Mol. Med. 1997, 75: 208-216.
	W	Mullins et al. Perspective Series: Molecular Medcine in genetically engineered animals. J. Clin. Invest. 1996, 98 (11), S37-S40.
	X	Seamark et al. Progress and emerging problems in livestock transgenesis: a summary perspective. Reprod. Fertil Dev. 1994, 6: 653-657.

*A copy of this reference is not being furnished with this Office action. (See MPEP § 707.05(a).)
Dates in MM-YYYY format are publication dates. Classifications may be US or foreign.

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FEE TRANSMITTAL for FY 2003

Effective 01/01/2003. Patent fees are subject to annual revision.

☑ Applicant claims small entity status. See 37 CFR 1.27

TOTAL AMOUNT OF PAYMENT ($) 55.00

Complete if Known	
Application Number	09/900,700
Filing Date	July 6, 2001
First Named Inventor	Keith D. Allen
Examiner Name	Peter Paras Jr.
Art Unit	1632
Attorney Docket No.	R-616

METHOD OF PAYMENT *(check all that apply)*

☐ Check ☐ Credit card ☐ Money Order ☐ Other ☐ None

☑ Deposit Account:

Deposit Account Number: 50-1271

Deposit Account Name: Deltagen, Inc.

The Commissioner is authorized to: *(check all that apply)*

☑ Charge fee(s) indicated below ☐ Credit any overpayments

☐ Charge any additional fee(s) during the pendency of this application

☐ Charge fee(s) indicated below, **except for the filing fee** to the above-identified deposit account.

FEE CALCULATION

1. BASIC FILING FEE

Large Entity Fee Code	Large Entity Fee ($)	Small Entity Fee Code	Small Entity Fee ($)	Fee Description	Fee Paid
1001	750	2001	375	Utility filing fee	
1002	330	2002	165	Design filing fee	
1003	520	2003	260	Plant filing fee	
1004	750	2004	375	Reissue filing fee	
1005	160	2005	80	Provisional filing fee	

SUBTOTAL (1) ($)

2. EXTRA CLAIM FEES FOR UTILITY AND REISSUE

		Extra Claims		Fee from below	Fee Paid
Total Claims		-20** =		X	=
Independent Claims		- 3** =		X	=
Multiple Dependent					=

Large Entity Fee Code	Large Entity Fee ($)	Small Entity Fee Code	Small Entity Fee ($)	Fee Description
1202	18	2202	9	Claims in excess of 20
1201	84	2201	42	Independent claims in excess of 3
1203	280	2203	140	Multiple dependent claim, if not paid
1204	84	2204	42	** Reissue independent claims over original patent
1205	18	2205	9	** Reissue claims in excess of 20 and over original patent

SUBTOTAL (2) ($)

**or number previously paid, if greater; For Reissues, see above

FEE CALCULATION (continued)

3. ADDITIONAL FEES

Large Entity Fee Code	Large Entity Fee ($)	Small Entity Fee Code	Small Entity Fee ($)	Fee Description	Fee Paid
1051	130	2051	65	Surcharge - late filing fee or oath	
1052	50	2052	25	Surcharge - late provisional filing fee or cover sheet	
1053	130	1053	130	Non-English specification	
1812	2,520	1812	2,520	For filing a request for *ex parte* reexamination	
1804	920*	1804	920*	Requesting publication of SIR prior to Examiner action	
1805	1,840*	1805	1,840*	Requesting publication of SIR after Examiner action	
1251	110	2251	55	Extension for reply within first month	55.00
1252	410	2252	205	Extension for reply within second month	
1253	930	2253	465	Extension for reply within third month	
1254	1,450	2254	725	Extension for reply within fourth month	
1255	1,970	2255	985	Extension for reply within fifth month	
1401	320	2401	160	Notice of Appeal	
1402	320	2402	160	Filing a brief in support of an appeal	
1403	280	2403	140	Request for oral hearing	
1451	1,510	1451	1,510	Petition to institute a public use proceeding	
1452	110	2452	55	Petition to revive - unavoidable	
1453	1,300	2453	650	Petition to revive - unintentional	
1501	1,300	2501	650	Utility issue fee (or reissue)	
1502	470	2502	235	Design issue fee	
1503	630	2503	315	Plant issue fee	
1460	130	1460	130	Petitions to the Commissioner	
1807	50	1807	50	Processing fee under 37 CFR 1.17(q)	
1806	180	1806	180	Submission of Information Disclosure Stmt	
8021	40	8021	40	Recording each patent assignment per property (times number of properties)	
1809	750	2809	375	Filing a submission after final rejection (37 CFR 1.129(a))	
1810	750	2810	375	For each additional invention to be examined (37 CFR 1.129(b))	
1801	750	2801	375	Request for Continued Examination (RCE)	
1802	900	1802	900	Request for expedited examination of a design application	

Other fee (specify) _____

*Reduced by Basic Filing Fee Paid

SUBTOTAL (3) ($) 55.00

SUBMITTED BY				(Complete *if applicable*)	
Name (Print/Type)	Aaron T. Hokamura	*Registration No. (Attorney/Agent)*	51,810	*Telephone* 650-569-5171	
Signature	Aaron Hok			*Date*	02/24/03

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